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SOUTH AFRICAN PLANT POISONS AND THEIR INVESTIGATION.

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SOUTH AFRICAN PLANT POISONS AND THEIR INVESTIGATION.

By Charles Frederick Juritz, M.A., D.Sc., F.I.C.

About nine years ago I had the privilege of reading, before the South African Philosophical Society, as the Royal Society of South Africa was then styled, a few notes on such work as had been carried on in the two Government Chemical laboratories under my charge, at Capetown and Grahamstown, in regard to toxic principles in some plants indigenous to the country.* I expressed a deep consciousness of the cursory character of the little work that had been done, and expressed the hope—a hope many times thereafter repeated in my annual reports—that definite provision might at length be made for systematic research with reference to the important subject of South African plant poisons. I regret the fact that the position is no better to-day than it was in 1905—that is to say, South Africa still remains as indifferent as in Pappe's day to the pharmacological possibilities of its almost inexhaustible flora. On the other hand, European institutions are realising the desirability of performing the investigations which we neglect, and for some years past large quantities of some of these very plants have been collected in this country at the instance of Directors of laboratories in England, and have been conveyed thither for investigation. Some of the results thus arrived at overseas, with regard to South African plant principles, have been mentioned in Dr. Marloth's last presidential address to the Cape Chemical Society,† and I shall therefore refrain from dwelling on them in the present paper, practically confining my attention to indicating what additional information has been gained in the Cape Government laboratories, subsequent to 1905, by simple continuance along the lines described in my former publication on the subject. The species mentioned in that publication were as follows:—

Amaryllidaceæ.

Buphane disticha Herb.

Polygonaceæ.

Polygonum tomentosum Willd.

Meliaceæ.

Trichilia Dregei E. Mey.

Apocynaceæ.

Acokanthera venenata Don.

Rauwolfia natalensis Sond.

* "Some notes regarding South African Pharmacology," in *Trans. S.A. Phil. Soc.*, **16**, 111-133.

† R. Marloth: "The chemistry of South African plants and plant products," (1913).

ASCLEPIADACEÆ.

Gomphocarpus sp.

Before proceeding further, let me express my acknowledgments to Dr. Marloth for correcting, in his address, the error in my previous paper of identifying the *Umjela* or Quinine tree with *Tabernæmontana ventricosa* Hochst. I had accepted the identity as correct on the authority of the Forest Department, and Sim's *Botanical observations on Forests of Eastern Pondoland* (page 28) refers to *Umjela,* or Quinine tree, as *Tabernæmontana ventricosa,* but it now appears that the Quinine tree examined in the laboratories was not *Tabernæmontana* but *Rauwolfia natalensis.**

In the present paper I propose to call attention to the following plants in particular, most of which have been under examination in the Cape laboratories—principally in the laboratory at Grahamstown, by Mr. J. Muller—during the last few years:—

CYCADACEÆ.

Encephalartos spp.

LILIACEÆ.

Bowiea volubilis Harv.

AMARYLLIDACEÆ.

Clivia nobilis Ldl.
Clivia miniata Regel.
Hæmanthus natalensis Pappe.
Hæmanthus puniceus L.
Buphane disticha Herb.

RANUNCULACEÆ.

Knowltonia bracteata Harv.

RUTACEÆ.

Xanthoxylon capense Harv.

MELIACEÆ.

Melia azederach L.

EUPHORBIACEÆ.

Euphorbia pugniformis Boiss.

CELASTRACEÆ.

Elæodendron croceum DC.

MELIANTHACEÆ.

Melianthus comosus Vahl.

EBENACEÆ.

Euclea lanceolata E. Mey.

* See also T. R. Sim: "The forests and forest flora of the Colony of the Cape of Good Hope" (1907), 270, where the *Umjela,* or quinine tree, is identified with *Rauwolfia natalensis.*

APOCYNACEÆ.

Acokanthera venenata Don.

COMPOSITÆ.

Helichrysum serpyllifolium Less.
Helichrysum sp.
Dimorphotheca sp.
Diplopappus asper Less.

CYCADACEÆ.

Some time during 1912 two Pondo lads, aged about twelve years, died at Tabankulu, it was supposed, from eating the fruit of a plant afterwards identified as *Encephalartos*. In the first instance, however, they were represented as having eaten "nuts." Dr. Lewis, in charge of the Grahamstown laboratory, expressed a desire to investigate those "nuts," and in December of the year mentioned specimens of the foliage, fruit, and roots of the shrub alleged to have caused death were received by him. It transpired that the articles eaten by the boys were termed *um-Gusa* nuts. The boys roasted and ate the "nuts," it was said, about mid-day, and began to vomit at dusk. After a time vomiting ceased: they then lay down, refusing food, but drank water with avidity. From their recumbent position they did not rise again, but died eighty hours later. A local herbalist said that the father of one of the boys had told him that his son had pains in the chest, and slight swelling on each side of his neck. Twenty-four hours before death he could hardly speak. Post-mortem examination showed that death was due to exhaustion, consequent upon ingestion of an irritant poison. The peritoneum contained blood-stained fluid; the stomach was much inflamed, and the contents blood-stained; the intestines were also acutely inflamed. Dr. Lewis submitted the seeds (of which the outer fleshy coat had been removed) to Dr. Schönland, who pronounced them seeds of *Encephalartos*, probably *E. villosus*, and suggested submission to Dr. Rattray. A cone submitted to Dr. Schönland at the same time was considered by him to belong to a *different* species of *Encephalartos*. The exuding gum of *Encephalartos* cones is stated by Dr. Becker, on his personal observation at the Kowie, to be eaten by Kaffir children and by birds. Mr. Tidmarsh informed Dr. Lewis that the *Encephalartos* family is well known under the name of "Hottentots' bread," and that the cones are considered edible. He had never heard of poisonous properties being ascribed to them. Dr. Rattray wrote that all his information pointed to the conclusion that the seeds of some species of *Encephalartos* are eaten by Natives. He added that Dr. Medley Wood had stated some years ago that he had formerly supposed Natives to be in the habit of eating the pith, but he had since discovered that it is the seeds that they eat. All early travellers, Dr. Rattray went on to say, refer to *Encephalartos* as Kaffir or Hottentot bread, some giving minute details of the manner of

preparing the pith. Local Natives had told him that the seeds are edible; in fact, he had himself eaten seeds of *Encephalartos Altensteinii*. He had also offered *Encephalartos* seeds to monkeys. They eat the outer reddish coating, but reject the seed with its stony covering. The outer fleshy coat of the seed was not unpleasant, but the seed itself was astringent, like acorns. On the other hand, *Macrozamia Moorei*, a small cycadaceous plant of Australia, whose genus is nearly allied to *Encephalartos*, is reputed to be very poisonous to stock, and to be the cause of fatal rickets in cattle. Measures are therefore being taken for its eradication. Dr. Rattray was of opinion that different species of *Encephalartos* may possess different properties. Later on, the actual specimens received by Dr. Lewis were submitted to Dr. Rattray, and were identified by him as *E. Frederici-Guilielmi* Lehm., which is common around Queenstown and Cathcart, and is traceable eastwards as far as Tsomo. After identification, Dr. Lewis procured a further supply from Dr. Becker, and was thus enabled to make a thorough investigation of the carpels and of the seeds, both ripe and unripe. No glucoside was obtained from any part of the flower. From immature seed coats alone Dr. Lewis obtained a very small quantity of an alkaloid, which, even in large doses, was not toxic either to guinea pigs or to white rats. The conclusion was therefore arrived at that, if the lads did die from eating the fruit, they must have consumed large quantities, and died from over-eating. On the other hand, this inference leaves the "ingestion of an irritant poison" mentioned in the *post-mortem* report unexplained.

Liliaceæ.

At Indwe two natives lost their lives in 1910 through the administration of indigenous plant decoctions by a "Kaffir doctor." The bulbs partaken of were found to contain an acrid resinous body, and the plant was easily indentified as *Bowiea volubilis* Harv. The acrid substance extracted from this plant was experimented with, and was found to act as a strong emetic and an irritant poison.

Whether it was an alkaloid or a glucoside could not be predicated with certainty, on account of the small quantity (about 30 grammes of the bulb, with shrivelled leafy stalk attached) available. The colour reactions produced by it, which were not very characteristic, were as follows:—

Concentrated sulphuric acid	Yellowish to reddish brown.
Sulphuric acid and potassium bichromate	Yellowish brown to emerald green.

A quantity weighing 15 grammes was used for these chemical tests, as some of the material had also to be reserved for physiological experiments.

To a young dog, aged about 16 months, a decoction representing nearly 12 grammes of the pounded fresh bulb was admin-

istered. Evident discomfort followed in 15 minutes, and in another 15 minutes emesis took place, recurring thereafter at intervals, with violent straining, increased salivation, very irregular pulse, general depression, refusal of all food, and apparently cramping pains in the intestines. Death occurred 32 hours after administration.

Post-mortem examination, performed at the Veterinary Institute, resulted as follows:—

Rigor mortis well marked. Blood dark, tarry, and badly coagulated. Lungs: Slight congestion and atelectic patches at apices and lower edges. Heart distended (diastole), filled with tarry, imperfectly coagulated blood. Petechiæ on endocardium. Stomach completely empty, slight congestion towards lower end. Pylorus slate-grey in colour. Acute congestion and inflammation of the duodenum, jejunum, ileum. Mucous membrane covered with a dirty grey croupous exudate. Small intestines quite empty. Small quantity of ingesta in cæcum. Spleen, liver, kidneys, and glands normal. Consider death due to some irritant poison.

A three-year-old sheep was dosed with about 14 grammes of bulbs of the same species, made into a similar decoction. For four days no noticeable symptoms were observed; then the animal refused food, and died a day later.

The *post-mortem* report of the Veterinary Institute was as follows:—

Blood quite coagulated, except in small quantity in chest cavity not completely coagulated. Left lung: Pneumonia. Right lung: Normal. Heart: Normal. Stomach: Full, patchy congestion. Second and third stomachs normal. Fourth stomach acutely inflamed. Mesentery glands all congested. Small gut empty, patches of congestion. Large gut: Small quantity of food in it, thickened and congested in places. Liver congested and friable. Kidneys congested. Death due to some irritant poison.

To a small dog, about six or seven months old, a dose of about 2¼ ounces of the actual medicine* given to the deceased was administered. Great depression ensued, but no other ill effects were perceptible. On the third day, therefore, an almost equal amount was again administered. This brought about slight salivation, and apparently griping pains. On the second day after the latter administration persistent emesis occurred, and on the following day the animal expired.

The stomachs of both the deceased Kaffirs were acutely inflamed, and in both cases chemical examination revealed traces of the resinous body found in the bulbs. These organs were practically empty. In one of them five small pieces of solder, consisting of lead and tin alloy, and weighing in all .7 of a

* The bottle contained 190 c.c. of the "medicine," the liquid being very turbid, and commencing to ferment. It held in suspension several fragments of vegetable matter, which, under the microscope, were found to be identical with sections made from the bulbs of *Bowica volubilis* Harv. The liquid was acid to litmus, and low in extractive matter. Chemical analysis showed the presence of the resinous body already spoken of, but to a very much smaller extent than anticipated, having seemingly been destroyed by the incipient fermentation to an appreciable degree.

gramme, were found. Portions of the livers of the two deceased were also found to contain traces of the resinous body.

At Stutterheim in 1911 a native had been charged with causing a woman's death by administering to her a decoction of indigenous bulbs. Specimen bulbs, alleged by the accused to be similar to that so administered, were identified as *Bowiea volubilis* Harv. These, too, were found to contain a very acrid resinous body, which, on being physiologically experimented with, proved to act as an irritant poison and strong emetic. From the stomach of the deceased a small quantity of a resinous body, similar to that above mentioned, was extracted, and a similar extract was also obtained from the deceased's liver.

Amaryllidaceæ.

The unsatisfactory nature of the evidence that is frequently all that can be supplied in connection with chemico-legal investigations was exemplified in connection with the death of a native female at Mqanduli in 1905. Along with the organs of the deceased woman there were received (*a*) a parcel of roots, and (*b*) portion of a plant. The latter had been dug up by the man accused of having caused the deceased's death, and was said by him to be called *Mayime*,* and to be identical with that from which he had prepared the fatal decoction. The former was said to have been the actual plant from which that fatal decoction had been prepared.

First of all the lining of the stomach was found to contain thirteen worms (*Ascaris lumbricoides*), from 3½ to 9½ inches in length, of which, strangely enough, no mention was made in the report on the *post-mortem* examination. The chemical tests applied to the various articles above mentioned resulted as follows, the active principle being in each case obtained by evaporation of a chloroform solution of an alkaline aqueous extract of the article examined:—

* Some years after the occurrence of this case I learnt, from the Rev. A. T. Bryant's article on "Zulu medicines and medicine-men," in the Annals of the Natal Museum, **2** [1], 45, 98, that the name *u-Mayime* is applied by the natives to *Clivia miniata* Regel, a circumstance which should certainly lead to an investigation of the latter plant.

Reagent Applied.	Stomach.	Root (*a*).	Plant (*b*).
Concentrated sulphuric acid.	Soluble with yellowish brown colour, turning pink to dirty green.	Soluble with bright yellow colour, turning dirty green evanescent.	Soluble with slight yellowish colour, turning pink to faint green.
Concentrated nitric acid.	Soluble with yellow colour, permanent.	Soluble without colour.	Soluble with yellow colour, permanent.
Vitali's reagent.	Dark brown.	Nil.	Very dark brown.
Fröhde's reagent.	Dirty green.	Yellowish brown, changing violet to blue; speedily evanescent.	Pale yellowish green.
Sulphuric acid and potassium bichromate.	Dark brown to green.	Yellowish green to violet.	Yellowish brown to emerald green.
Sulphuric acid and nitric acid.	Soluble with pale yellow colour.	Successively bright yellow, pink, violet, lake, fading to yellowish.	Soluble with very bright yellow colour.
Bromine water.	Nil.	Nil.	
Taste.		Slightly bitter.	Very bitter.
Solubility.		Soluble in very dilute acids, in alcohol, chloroform, and ether.	Soluble in dilute acids, in alcohol, chloroform, and in mixture of ether and chloroform.

It was quite obvious that the plants (*a*) and (*b*) were different. It was impossible to identify the latter, although it closely resembled *Clivia nobilis* in certain particulars, but differed from it essentially in others. From a freshly cut surface of the plant a milky and somewhat sticky liquid exuded, possessing an acid reaction and a very bitter and extremely nauseating taste. The plant contained 2 per cent. of a very acrid yellow resinous substance, as well as a non-crystallisable, apparently glucosidal body. It was from this that the reactions tabulated in the last column above were obtained. Physiologically it produced—beyond a slightly depressant effect—no serious inconvenience when administered to a rabbit, while in the case of a dog, a decoction from a quarter of an ounce of the plant produced, within ten minutes, emesis accompanied by severe straining. Signs of general constitutional disturbance continued for five hours, after which complete recovery set in. Some days later the administration of a slightly increased dose produced more marked effects, and it is probable that in the case of a person suffering from a serious chronic complaint, as the deceased obviously was, fatal consequences may ensue upon the administration of so powerful an emetic. It is unfortunate that, up to the present, this plant has not been identified, but it is clear that it was quite distinct from the root actually partaken of by the deceased. The latter apparently contained a gucoside, the physiological effects of which, however, it was impossible to trace, owing to the small quantity available. It is quite possible that the deceased had partaken of a decoction derived from a plant similar to that dug up by the accused, but the reactions of the stomach extract did not seem to show the presence of the root from which, it is said, the actual fatal decoction had been prepared. The whole connection therefore remains unsatisfactory, and it would be worth while to pursue the thread afforded by the plant *Mayime,* which the accused himself subsequently dug up.

As regards *Clivia nobilis,* it may be said that in connection with the death of a little Kaffir girl at Willowvale in 1904, a specimen of that plant was received, and a glucoside normal thereto was discovered in the contents of the deceased's stomach. From physiological experiments made with the plant, the conclusion was drawn that, unless very excessive doses were administered, no apparent effect was produced; a fairly large quantity of a strong decoction acting merely as a mild emetic. The plant mentioned belongs to a group very closely related to the *Hæmanthus* family, of which several members have been used as emetics and purgatives. In this particular case the child was only 15 months old, and had been sickly from its birth.

In connection with the death of a native woman at Idutywa in 1905, a portion of a bulb called by the natives *Gobisi* was submitted, together with the statement that the other part had been administered to the deceased shortly before her death by the

person charged with culpable homicide. From the organs of the deceased chloroform extracted, from an alkaline aqueous solution, a slightly yellowish non-crystallisable substance, which yielded the following reactions:—

Solubility.	Very slightly soluble in water; very soluble in chloroform, ether, alcohol, and dilute acids.
Action on litmus paper.	Alkaline.
Taste.	A few scales of the residue produce primarily a sharp burning sensation when placed on the tongue, speedily disappearing, and being succeeded by a somewhat persistent bitter taste, increasing salivation, and producing distinct nausea.
Concentrated sulphuric acid.	Soluble with pinkish colour.
Concentrated sulphuric acid with potassium dichromate.	Nil.
Gold chloride.	Yellow curdy precipitate.
Platinum chloride.	Faint whitish precipitate.
Concentrated nitric acid.	Readily soluble, with light yellow colour.
Phosphomolybdic acid.	White curdy precipitate, insoluble in excess.
Phosphotungstic acid.	White curdy precipitate, insoluble in excess.
Vitali's reagent.	Dark reddish-brown coloration.
Fröhde's reagent.	Pink; more prominent than with sulphuric acid only.
Picric acid.	White curdy precipitate.
Iodo-potassium iodide	Dark red cloudy precipitate.

From the bulb *Gobisi,* on treatment for a week with acidulated alcohol, and subsequent evaporation and extraction with chloroform, yellow non-crystallisable scales were obtained in quantity amounting to .168 per cent. of the bulb, and possessing properties similar to the substance extracted from the stomach of the deceased.

It was quite impossible to identify the plant botanically from such a specimen as that was, and so the Resident Magistrate of Idutywa subsequently furnished an entire specimen of a bulb, with leafing stalk attached, and stated to be identical with that administered by the accused to the deceased. This bulb was planted, by permission of the Director of the Albany Museum, in the grounds adjoining that institution, and on flowering it was identified as *Hœmanthus natalensis* Pappe. This particular bulb was also analysed, and yielded reactions in all respects identical with those obtained from the deceased's organs, and from the

portions of the bulb *Gobisi* previously received. It appeared evident, therefore, that, whether death was caused by her having done so or not, the woman had, shortly before her decease, partaken of *Hæmanthus natalensis*.

Physiological experiments were also made with portions of the bulb of that plant. A decoction equivalent to 14 grammes of the fresh bulb, administered to a five months old dog, produced severe emesis within twelve minutes, and symptoms of malaise continued for about three hours, after which complete recovery set it. Two days later a larger quantity—equivalent to 32 grammes of the bulb—was administered to the same animal, with a repetition of the former results. Finally, one-ninth of a grain of the active principle itself was administered to the dog, and precisely similar results were again observed. Doses of from six to ten grammes of the bulb, given in the form of a decoction, were next administered to rabbits without any apparent result. One animal died within six days, but without obvious connection with the previous administration of the bulb decoction, as no signs of ill-health occurred in an animal treated with larger doses. On a strong vigorous mouse a dose of one-twelfth of a grain of the active principle produced no apparent effect whatever.

The conclusion arrived at was that the plant is not directly poisonous, but that the violent straining, accompanying the emesis produced by the bulb, acted fatally, a tendency which would appear possible in subjects suffering from heart disease or some other serious organic complaint.

Bryant refers to the fact that decoctions of the roots of *H. natalensis* are used in cases of bronchitic and other coughs as an emetic to remove the "wild beast's hair," the presence of which in the air passages of the patient is supposed to cause the cough.

In 1909, in connection with the poisoning of a native woman in the Albany Division, a Kaffir "doctor" had been charged with culpable homicide, and a small piece of the bulb stated to have caused death, weighing less than 40 grains, was submitted for analysis. The specimen was destitute of leaves, and gave no clue as to the plant or species to which it belonged. After a lapse of about two months, the Analyst in charge of the Government Laboratory at Grahamstown succeeded in procuring specimens of certain bulbs growing in that district, which on examination proved to be identical with the piece of bulb previously handed in, both microscopically and chemically. The bulbs so procured belonged to the genus *Hæmanthus*, and, as it was not then the flowering season for that plant, it was difficult to tell whether the particular specimen was *H. puniceus* or *H. magnificus*—an illustration of the advantage which even the course of justice would reap by making the study of the poisonous plants of the country entirely independent of the momentary requirements of legal proceedings. At all events, the difference between the two plants named is so slight that, when grown under varied conditions, it is sometimes impos-

sible to distinguish between them. Specimens of *Hæmanthus* were found growing in a wooded kloof near Collingham, in the Albany Division, and, after considerable delay, a sufficient number of these bulbs for analytical purposes was procured from the above-mentioned locality.

From those bulbs a fair amount of resin was extracted, as well as an active principle, which in every respect answered to the general tests for alkaloids, and gave the following characteristic reactions:—

Colour.	Pale yellow.
Solubility.	Difficultly soluble in cold water; more readily soluble on heating.
Hydrochloric acid.	Very soluble.
Nitric acid.	Soluble, with a canary-yellow colour.
Sulphuric acid.	Bright yellow, changing to brown and dirty greenish yellow.
Sulphuric acid and Potassium dichromate.	Yellowish-brown, changing to dark brown.
Wagner's reagent.	Thick cloudy white precipitate.
Dragendorff's reagent.	Vermilion-coloured precipitate.
Sonnenschein's reagent.	Thick curdy white precipitate.
Picric acid.	Yellow precipitate.

The active principle therefore seemed to be an alkaloid new to science. The bulb, when fresh, contained 70 per cent. of water, and from 20 to 24 per cent. of starch, the remainder consisting of resin, colouring matters, etc., and the alkaloid referred to above. From 100 grammes of the dried bulbs nearly 15 grains of the alkaloid were extracted, so that the fresh bulb would contain approximately from .25 to .30 per cent. of this active principle.

In order to ascertain whether the plant had any pronounced toxic properties, a quantity of the fresh bulb, weighing 35 grammes, was made into a decoction, and administered to a healthy full-grown sheep, A. It developed no marked symptoms, but went off its feed, and died in about 66 hours. A decoction was then made of another portion of fresh bulb, 45 grammes by weight: this was given to a second healthy full-grown sheep, B. Here, too, no typical symptoms ensued, but the animal refused food, and died in 55 hours. *Post-mortem* examinations were made by the Director of the Veterinary Institute, who stated in his report that

> The animals died quietly; no sign of purging; slight discharge of mucus from one nostril in the case of sheep B. In the fourth stomach, suggestion of ecchymosis; the organ contained a quantity of fluid. Mucous membrane inflamed; small petechiæ scattered over surface. . . . Heart engorged, very much dilated. Kidneys: marked congestion, particularly so in sheep B.

To a healthy guinea-pig 2½ grains of the purified alkaloid were administered; the animal died in little over four hours.

It was obvious, then, that the bulb, stated to have been used by the accused for medicinal purposes, contained an alkaloid possessing toxic properties so marked that it is easy to understand fatal results following upon an overdose of the drug.

After the investigation had proceeded thus far, the stomach and liver of the deceased, which had been preserved all this while, until definite conclusions could be reached with respect to the supposed poisonous nature and chemical properties of the bulb, were examined. Upon extraction by the usual processes, a residue was finally obtained, which, after repeated purification, and treatment with the reagents for alkaloids mentioned above, gave reactions in every respect similar to those of the alkaloid extracted from the *Hæmanthus* bulb. The amount extracted from the stomach and liver was, however, too small to determine quantitatively, or to allow of physiological tests being made, but from the chemical reactions and colour tests described, it was, with a high degree of probability, identical with the alkaloid found in the bulb, thus confirming the opinion that the woman died from the effects of the bulb administered to her.

Four months were occupied in the endeavours to establish all the connecting links in this chain: had the toxic properties of this plant been previously studied systematically, and, for its own sake, as part of that research into the nature and characteristics of the plant drugs and plant poisons of South Africa that I have been urging these many years past, it is probable that the scientific links in the chain of evidence would have been complete in scarcely more days than they actually required months.

A case which occurred at Nqamakwe in 1911 involved the chemical examination of no less than twelve articles, namely, the organs of the deceased infant, four native drugs, two samples of bark, a bulb, a dried root, a teaspoon, the "medicine" administered to the child, and some of the brandy used for preserving the organs. From the viscera was extracted a very small quantity of an active principle resembling the alkaloid of *Hæmanthus puniceus* or *magnificus*—the same plant which had caused the prolonged search above commented on. The amount extracted from the organs was, however, too small to permit of its quantity being estimated, or of physiological experiments being made therewith. Pieces of the tuber said to have been administered to the infant, on microscopic examination, revealed its similarity to *Hæmanthus*. Ten grains, made into an infusion with water, and administered to a kitten aged about five weeks, caused speedy vomiting, and death in twelve hours. Only 4½ grains of the material were left after this physiological experiment, and, as the fresh substance contains, in 1,000 parts, barely 2½ of the active principle, it is not astonishing that chemical

tests showed only faintly the characteristic *Hæmanthus* reactions. An entire specimen of the bulb, with leaves attached, but no flowers, accompanied the other exhibits in this case, and, on chemical examination, it was found to yield the following reactions:—

Colour.	Pale Yellow.
Solubility.	Difficultly in cold water; more readily on heating.
Hydrochloric acid.	Soluble, with a canary colour.
Sulphuric acid.	Bright yellow to brown and dirty greenish yellow.
Sulphuric acid and potassium dichromate.	Yellowish brown to dark brown.
Wagner's reagent.	Thick cloudy white precipitate.
Dragendorff's reagent.	Vermilion-coloured precipitate.
Sonnenschein's reagent.	Thick curdy white precipitate.
Picric acid.	Yellow precipitate.

These reactions are similar to those already detailed above. Similar reactions were also given by an active principle extracted from the child's viscera. Other articles examined in this connection were: (1) Bark known as *Sidivadiva,* which could not be identified botanically, but was apparently non-toxic; (2) bark described at *Mtshekisana,* also non-toxic and incapable of identification; but the same native name has been applied to *Diplopappus asper* Less. (*Aster hispidus* Bak.), in which an acrid poisonous body has been found, possessing, in even moderate doses, strong emetic properties*; (3) a root called by the Natives *Makubala,* containing traces of a bitter principle, similar to the glucoside acokantherin, but in quanties too small to experiment with; (4) the leafing stalks and stems of a plant called *Sihlahla,* belonging to the Labiatæ, and possibly *Teucrium riparium* Hochst, but possessing no marked toxic properties.

About the middle of 1913 a native woman at Port St. John's died, it was supposed, from the effects of some poison inserted either *per vaginam* or *per rectum.* When the *post-mortem* examination was held, five days after death, decomposition had only just begun. The stomach and colon of the deceased were submitted to analytical tests, and from one-third of these organs there was extracted .8 milligramme of an alkaloid which was subsequently definitely identified as Buphanine, one of the alkaloidal principles of the poisonous plant *Buphane disticha,* commonly known in this country as "Giftbol," and by the Kaffirs as *in-Cwadi.* This is one of the plants that has been investigated overseas, and the physiological action of Buphanine is described by Tutin as follows:—

Buphanine produces physiological effects similar to those of hyoscine and hyoscyamine, particularly the former, but its action is weaker and less

* See post, page 23.

lasting than either of the last-mentioned bases. It is a mydriatic, and paralyses the salivary secretions and the vagus endings in the heart, and causes death by respiratory failure of central origin.*

This plant was referred to under the name *Buphane toxicaria* on page 121 of my previous paper on South African Pharmacology, and details were then given of colour reactions produced by the active principles contained in it.

Ranunculaceæ.

A native girl was declared to have been poisoned at Bizana in 1910, and in that connection two indigenous plants were submitted for examination. One of these, known by the Natives as *Montuza,* was identified as *Knowltonia bracteata* Harv.

The specimen received was too small to permit of the application of exhaustive chemical or physiological tests.

The genus *Knowltonia* is known to be toxic, and certain species—the commonest of which are *K. vesicatoria* and *K. rigida* —are extremely acrid, and are used as popular remedies for rheumatism and lumbago. According to Pappe,† the acrid bruised leaves raise effective blisters when applied to the skin.

From the quantity of the plant supplied a fairly large amount of resinous body was extracted, which, after separation and purification, gave the following very characteristic reactions:—

Concentrated sulphuric acid.	Brown to yellowish brown.
Sulphuric acid and potassium dichromate.	Pale reddish pink, changing after an hour to Vandyke brown.
Nitric acid.	Pale yellow.
Alkaloidal precipitants.	Nil.
Alcohol.	Soluble.
Water.	Insoluble.
Acidulated water.	Insoluble.

Nine grammes of the plant material were made into a decoction, and administered to a healthy vigorous dog, without any apparent effect whatever.

Rutaceæ.

During the year 1906 cantharidin was found in the stomach of a Native who died at Libode, and also in a powder stated to have been administered to the deceased, but the chief interest of this case just here centres in the fact that it led to tests being made with the root and leaves of a plant said to be known as *um-Nungamabele* (or Knobwood), also declared to have been adminis-

* Tutin: "The constituents of the bulb *Buphane disticha*": Wellcome Research Laboratories, London. See also *Rept. S.A. Assn. for Adv. of Science,* Capetown (1910), 385.

† Flora capensis med. prod" (1857), 1. See also Harvey and Sonder: "Flora capensis," 1, 4.

tered to the deceased. The root and branches were identified as belonging to *Xanthoxylon capense* Harv., which Smith, in his "South African Materia Medica,"* also identifies with *um-Nungamabele,* but to which Bryant gives the native name of *um-Nungwane.* At a later stage reference will be made to a plant submitted under the name *um-Nungamabele,* and found to be *Rhus discolor* E. Mey. The Knobwood is used by the Kaffirs as a disinfectant of miltziek meat, which is either boiled with the leaves or else roasted, and, after being partaken of, is immediately followed by an infusion of the Knobwood leaves. This seems to controvert any idea as to the plant being poisonous.

In investigating this particular case, the root and twigs were powdered and submitted to the Stas-Otto and Dragendorff methods of analysis. A fairly large proportion of a resinous body was extracted, together with tannins and traces of a yellow colouring matter, for which no characteristic tests were ascertained. There were apparently no active principles present that could be denominated poisonous. An aqueous decoction equivalent to 11 grammes of the powdered root and leaves, administered to a strong healthy dog, aged about eleven months, produced no visible effect. Further examination of the powder, whereof the deceased had partaken, showed that it consisted of the ground root of the Knobwood, mixed with crushed cantharides, and it was evidently the latter and not the former that caused death.

The association of *Xanthoxylon capense* with cantharides in this case is somewhat curious, in view of the circumstances related by Bryant. It appears that an infusion of the roots of *X. capense* is used as one of the remedies for what are declared to be intestinal parasites known by the Zulus as *i-Khambi.* This is the imago of a beetle with greenish-black elytra, almost identical in appearance with the ordinary dung beetle (*Aphodius marginicollis* Har.).

It may also be of interest to mention here that, in connection with a case at Mount Frere in 1908, together with the organs of the deceased, there was submitted for examination a goat's horn containing drugs reported to have been administered to the deceased by the "Kaffir doctor" who treated him. In this horn was found a powder consisting of crushed specimens of the South African blistering fly, *Mylabris bifasciata,* which is known to yield more than double the proportion of cantharidin procurable from the Spanish fly, *Cantharis vesicatoria.* In this powder nearly 2 per cent. of cantharidin was found, together with 7 per cent. of extractive matter possessing blistering properties in a high degree. In the stomach and intestines of the deceased, fragments of the elytra or wing-cases of the *Mylabris* were also found, but neither in the stomach nor in the intestines could any cantharidin be discovered by chemical tests. On the

* 3rd ed. (1895), 44, 58, and 126.

other hand, a non-alkaloidal body, apparently a glucoside, was found, both in the stomach and in the small intestine. About this latter substance there was apparently nothing toxic, but its presence proved that other drugs than the insect had also been administered to the deceased. Evidence showed that the quantity of cantharidin swallowed by him must have been at least 30 or 40 grains—*i.e.*, it exceeded the minimum fatal dose.

Meliaceæ.

A fatal case of poisoning with the berries of the Cape Syringa tree (*Melia azederach*) occurred at Kingwilliamstown in 1910. Some tests were accordingly made in the Grahamstown Laboratory. Twenty-five berries, powdered and subsequently boiled to a thick gruel, were administered to a healthy sheep without any effect whatever. Seventy-five berries, similarly prepared, produced no noticeable effect on a healthy calf, nor did fifty-one berries on a young healthy dog. Whatever poisonous principle these berries may contain, therefore, is apparently due to a glucoside, inasmuch as it is evidently destroyed by boiling.

Euphorbiaceæ.

No cases of *Euphorbia* poisoning have come under the notice of the Cape Government Analysts, but, as it may be of interest to add to these notes some of the results obtained by Prof. Hahn, during the time that he was retained as analyst by the Law Department of the Cape Colony, extracts embodying such results have been made from the records of that Department. Amongst these was the case of a Native who died at Middelburg early in the year 1900, from the effects, it was supposed, of *Euphorbia* poisoning, a decoction of *Euphorbia* having been drunk by him. The plants alleged to have caused death, and submitted to Prof. Hahn for analysis, along with the deceased's organs, proved to be *Euphorbia pugniformis* Boiss., known to the Natives as *in-Kamamasane*. It is much used by Natives on account of its action as an irritant aperient. When employed in large quantities it acts as a virulent irritant poison.* From these plants Dr. Hahn extracted a yellow resinous substance, euphorbin, a powerful vesicant, drawing blisters when applied to the skin. In all his practice, Dr. Hahn declared, he had never before observed the effects of an irritant poison in so pronounced a manner. The greater portion of the inner walls of the stomach, he said, were dark and bluish black, indicating a high degree of inflammation. The upper epidermis was quite loose at the dark portion of the inner walls, showing that actual blisters must have been drawn at the time when death took place. On analysing the stomach and contents, Prof. Hahn found 5½ grains of euphorbin. The inner coating of the intestines he also

* Smith: "South African Materia Medica": 3rd ed. (1895), 119, 137.

found to be highly inflamed, and in these ¾ of a grain of euphorbin was found.

In another case, which occurred at Mount Fletcher in 1901, the deceased had partaken of a preparation of a species of *Euphorbia*. From succulent portions of the plant submitted to Dr. Hahn he extracted 36.7 per cent of *Euphorbia* resin. Dr. Hahn described this as "a powerful irritant poison, acting as an emetic, and producing bloody discharges and rapid and acute inflammation of the stomach and intestines." Two grains of this resin caused the death of a guinea-pig. From the deceased's stomach and its contents Dr. Hahn extracted 3½ grains of *Euphorbia* resin of the same kind, and possessing the same properties as that which he had extracted directly from the plant.

Celastraceæ and Ebenaceæ.

In the Peddie Division, during June or July, 1912, a Native died, it was believed, from the effects of taking roots of either *Elæodendron croceum* or *Euclea lanceolata*. No analyses were made in connection with this case, but with regard to the first-named (called in Dutch "Saffraan hout," and by the Natives *um-Bomvane*, from its red roots), Smith states that it was employed in trial by ordeal, and that "it is said that all who drank the decoction died."*

As to *Euclea lanceolata*, the same authority says that "the bark of the roots is purgative: a decoction is employed, and its action is said to be rapid."

I am not aware that any analyses of these materials have at any time been made, but, having regard to their declared strong emetic and purgative properties, it is probable that, at all events, in the case of persons of weak constitution, their action may be fatal.

Bryant points out that all species of *Euclea* are strongly cathartic, possibly even injuriously so, inasmuch as they are said frequently to draw blood. Especially is such the case with *E. lanceolata*.

Melianthaceæ.

The death of a Native at Whittlesea, in 1912, after administration of some Kaffir medicine, was apparently due to the mixture having been prepared from the leaves of *Melianthus comosus* Vahl., commonly known as "Truytje-roer-my-niet." The stomach of the deceased yielded very distinct traces of the presence of an active plant principle, which Mr. Muller found to be identical with that afterwards extracted by him from the medicine whereof a portion had been partaken by the man. This active principle, as found in that medicine, gave the following reactions:—

* Smith: "South African Materia Medica," 3rd ed. (1895), 178

Water.	Almost insoluble.
Acidulated water.	Soluble with difficulty.
Alcohol.	Readily soluble.
Ether.	Slightly soluble.
Chloroform.	Readily soluble.
Picric acid.	Pale yellow, curdy, non-crystalline precipitate.
Mercuric chloride.	Nil.
Schulze's reagent.	Light cherry-red to pink.
Fröhde's reagent.	Cherry-red to brick-red to reddish-brown, finally fading to bluish-green.
Gold chloride.	Nil.
Platinum chloride.	Nil.

Associated with this medicine were some plant roots, which seemed to have been crushed, and had apparently been boiled. These yielded an active principle also responding to the above reactions, and such was also the case with some apparently untreated roots and stems. A specimen of leaf, stated to be from the plant whence these roots had been derived and the medicine prepared, was identified as *Melianthus comosus* Vahl. Specimens of that entire plant were therefore procured from Whittlesea, and the leaves, stems, and roots thereof were separately dried and submitted to analysis. The dried roots contained .065 per cent. of the active principle already alluded to. In the stems the quantity was very much less, and in the leaves there was practically none of it. Attempts to crystallise this active principle proved unsuccessful. It yielded the following reactions:—

Water	Insoluble
Alcohol	Readily soluble
Ether	Difficultly soluble
Chloroform	Readily soluble
Picric Acid	Pale yellow, curdy, non-crystalline precipitate
Phospho-molybdic acid	Heavy white curdy precipitate
Mercuric chloride	Nil
Iodine in potassium iodide	Heavy dark brown precipitate
Schulze's reagent (antimonic chloride in phosphoric acid)	Cherry-red to pale pink
Concentrated sulphuric acid	Bright-red to brick-red, to chocolate-brown, to yellowish-brown
Concentrated sulphuric acid and potassium dichromate	Bright-red to yellow-brown to greenish-blue
Concentrated sulphuric acid and nitric acid fumes	Bright-red to brick-red, rapidly fading to light pink
Concentrated nitric acid	Dissolves without colour

Vitali's test	Yellowish-brown to dark brown
Fröhde's reagent	Cherry-red to brick-red, to reddish-brown, to bluish-green
Potassium mercury iodide	Nil
Potassium cadmium iodide	White precipitate
Potassium zinc iodide	Nil
Gold chloride	Nil
Platinum chloride	Nil
Sodium hydroxide	White precipitate, insoluble in excess, but soluble in alcohol

It is therefore reasonable to conclude that the drug administered to the deceased, and detected in the stomach contents, was the same as that found in the medicine prepared from the roots of *Melianthus comosus,* but to confirm the conclusions, by establishing the toxic nature of these roots, and, incidentally, the relative harmlessness of the stems and leaves, the following physiological experiments were performed. Two healthy guinea-pigs were fed with leaves which had been soaked in a decoction of the leaves and roots, but with no apparent ill effects. They were subsequently given 1 c.c. of a 5% infusion of the stems, without perceptible effect. To a healthy young dog, aged about ten months, 5 c.c. of a 6% decoction of the leaves and stems was administered. Within 30 minutes vomiting commenced. There was extreme salivation and lassitude, vomiting being repeated with severe straining. The dog's temperature was 104° F., and on the following morning 103.4° F. For 24 hours it refused food, and then gradual recovery set in, and on the third day the effects of the dosing were hardly noticeable. Several weeks later 10 c.c. of a 10% aqueous infusion of the crushed roots of this plant were injected subcutaneously in the groin of the same dog. Lassitude, loss of appetite, and vomiting occurred an hour later, and before another hour had passed there was some twitching of the limbs, the eyes were half closed, and lassitude increased. The dog was very quiet, refusing all food during the next two days, although it seemed to recover partially at intervals. It died during the night of the third day, or approximately sixty hours after injection. The Acting Government Pathologist made a *post-mortem* examination of the dog, and reported:

> Local necrosis of subcutaneous tissues at sites of inoculations. Congestion of great glands and internal viscera. Right ventricle of heart distended with *post-mortem* clot in diastole. Left ventricle in a condition of systole and empty.

It appears therefore that *Melianthus cosmosus* possesses very toxic properties, more especially in the roots; further, that an infusion made from the latter acts as a violent depressant, producing emesis, and also exerts a marked cardiac action, resulting fatally when administered in large doses. Pappe, in

his *Florœ Capensis Medicœ Prodromus,** refers to the use of a decoction of the leaves for necrosis, foul ulcers, etc., but makes no mention of employment of the roots of the plant, in which it is apparent that the toxic properties chiefly reside.

Apocynaceae.

Not much further progress has been made with regard to the investigation of the acocanthera poison, which was dealt with rather fully in my previous paper.† There were, however, a few cases of undoubted poisoning by *Acokanthera venenata.*

Towards the end of 1904 a case of a Kaffir girl about 15 years old, who had died at Cala, came under the notice of Mr. J. Muller, Assistant Analyst in charge of the Grahamstown Laboratory. Accompanying the organs of the deceased was a piece of wood, from which the poisonous glucoside yielding the characteristic *Acokanthera* reactions was easily extracted. From the stomach and its contents a very small quantity of an uncrystallisable residue, yielding the same characteristic reactions, was isolated. The quantity was too small to determine accurately.

In 1905, in connection with the death of a male native at Fort Beaufort, and of a female at Cala, the reactions previously described by me were in both cases obtained from the stomachs and contents of the deceased. It should be remembered that about one-third of a grain of the pure *Acokanthera* poison is enough to cause death to an average human being. In the Fort Beaufort case the quantity of the active principle recovered from the organs was very minute: it was probable that some of it had been lost by vomiting, and some absorbed into the system, for the drug is obviously one capable of very rapid absorption. In the Cala case, too, the amount of glucoside extracted from the organs was too small to determine quantitatively, but amongst the collection of plants submitted together with those organs the *Acokanthera* was represented by specimens of the wood, and of twigs with the leaves attached. In both cases the characteristic reactions with concentrated sulphuric acid were observed: I refer particularly to the colour test, which is of a most pronounced character; yellow, changing first to pink, then brick-red, and finally becoming violet.

In a case which occurred at St. John's in 1908, a Kaffir woman died after partaking of the roots of *Acokanthera venenata.* On this occasion, however, the *Acokanthera* was associated with *Cyanthula cappulacea,* or, as it is called by the Kaffirs, *u-Bulawu.*‡ The latter, which the woman had previously taken without ill-effect, is easily distinguished from

* 2nd ed. (1857), 6.

† Notes regarding S.A. Pharmacology," *Trans. S.A. Phil. Soc.* (1905), 16 [2], 123-128. *Vide* also C. G. H. Senior Analyst's Annual Reports (1902), 64-69; (1903), 60-63.

‡ According to Smith (see S.A. Materia Medica, p. 42) the name *ili-Bulawa* is applied by the natives to *Sebœa crassulæfolia* Schl.

Acokanthera in that it does not possess a bitter taste, and, when pounded up with some water, after having been freshly collected, yields a liquid alkaline to litmus, and produces a lather like soap. No toxic qualities were discoverable in the *Cyanthula,* but from the stomach and stomach contents a glucosidal body was extracted, which exhibited the following characteristic reactions:—

Fröhde's reagent	Yellowish-brown, changing into brownish-green
Concentrated sulphuric acid	Yellow, changing (1) to pink, (2) brick-red, and finally violet
Concentrated sulphuric acid and potassium dichromate	Emerald green, changing into clear blue

It was in my paper read in 1905 that I first dwelt at length on the cases in which *Acokanthera* poisoning had been enquired into in the Cape Government laboratories. On looking through the Law Department files it was found that, five years previously, a similar case had been submitted to Dr. Hahn. From a sample of bark (1¼oz.), which resembled that of *Acokanthera venenata,* he extracted, in addition to a large quantity of tannin, an alkaloid, which he found to resemble brucine in its chemical characters. From 300 grains of the bark he extracted 7½ grains of this alkaloid, of which a little less than one grain proved fatal to animals experimented upon. An infusion prepared from 40 grains of the bark was given to a guinea-pig, which died within half an hour. Dr. Hahn also found the bark to contain 18 per cent. of tannin, which, when taken in concentrated solution, acts as an irritant poison. In this instance Dr. Hahn found ¾ of a grain of the alkaloid above mentioned in the intestines of the deceased. The stomach and intestines also showed lesions due to the action of an irritant poison, and were hard and stiff, a condition which he ascribed to the tannin in the bark.

Compositae.

Two persons died at Eerste River in 1912 shortly after having partaken of "tea," which they had brewed on the veld from the stems and leaves of a particular bush. The organs of the deceased, and other food eaten by them, were found to be free from poisonous principles, and the twigs and leaves, stated to belong to the bush from which the "tea" was made, were identified as *Helichrysum serpyllifolium,* a plant not known to possess any toxic properties.

In connection with the death of a native woman at Elliotdale in 1908, a root and some leaves were submitted, of which it was impossible absolutely to establish the identity. Another root of similar appearance was procured by the analyst, and pronounced to be *Helichrysum* sp. This was tested chemically and physiologically. It contained 10 per cent. of an acrid resinous body, which yielded the following reactions:—

Sulphuric acid.	Cherry-red; very lasting; changing to lake in three hours.
Sulphuric acid and potassium dichromate.	Greenish-yellow, changing to pale pink.
Nitric acid.	Soluble; pale orange.
Fröhde's reagent.	Purplish; changing to dark greenish-black.
Water.	Slightly soluble.
Alcohol.	Soluble; precipitated on dilution.
Ether.	Soluble.
Reaction to litmus.	Distinctly acid.

On heating with water a peculiar nauseating odour was observed.

The specimen originally submitted gave the same reactions, so that, although botanically the two plants could not be pronounced identical, the chemical tests went to show their identity.

The plants were physiologically experimented with on a dog and two guinea-pigs; in the case of the dog, vomiting and slight purging ensued within half an hour, but these rapidly passed away, and in three hours the animal was in perfect health. One of the guinea-pigs was apparently not inconvenienced in the slightest; the other became restless and uneasy for half an hour, and then recovered normal health. The plant evidently is a depressant, and acts as an emetic and a slight purgative, but with no striking toxic effect. Nothing similar to this plant principle was discoverable in the stomach of the deceased woman; unless, therefore, it had been removed by vomiting, death was apparently due to some other cause.

Analyses have been made of two samples of the herb known as "Bietouw" (*Dimorphotheca*): one of these was obtained from Barkly East, and the other from Herschel. In both samples small quantities of hydrocyanic acid were found, amounting, in the Barkly East sample, to .0068 per cent., and in the other to .0077 per cent. In the former case the hydrocyanic acid was obtained by distillation, and in the latter by simple extraction, in both instances with dilute acid. Apropos of this, it may be mentioned that, according to Prof. Cavers,* hydrocyanic acid is found in the young leaves of sorghum. The amount, he says, is large enough in a dry season, or where the plants are grown on a dry soil, to be dangerous to cattle.

In connection with the Bizana poisoning case already alluded to, one of the two plants submitted, as before stated, was locally known as *Nozixekana*, and in Elliotdale as *Mtshekisana*, and was identified as *Diplopappus asper* Less. (*Aster hispidus* Bak.). The other was known by the Natives as *Montuza*, and was iden-

* *Knowledge* (1910), 7, 481.

tified as *Knowltonia bracteata*.* The roots and tubers of the *Diplopappus* contain a very acrid resinous body, amounting in the air-dried material to nearly .19 per cent. This active principle was found to yield the following chemical reactions:—

Concentrated sulphuric acid.	Cherry-red, changing to lake in three hours.
Sulphuric acid and potassium dichromate.	Greenish yellow to pale pink.
Nitric acid.	Soluble; pale orange.
Fröhde's reagent.	Purplish to dark greenish-black.
Water.	Slightly soluble.
Alcohol.	Soluble; deposits on dilution.
Ether.	Soluble.
Litmus paper.	Distinctly acid.

Apparently this was the same species of plant that was mentioned above as a species of *Helichrysum*.

Unidentified Plants.

Many cases arise in which it is an utter impossibility to identify the plants suspected or ascertained to be the cause of death or illness. Some of them may react with certain test reagents in the most striking manner, but such reactions bring us no nearer to botanical identification of the plant substances which produce them, for, as Dr. Marloth pointed out last year, in his Presidential address to the Cape Chemical Society,† there are over 12,000 species of flowering plants in South Africa, or over six times the number in the British Isles, and very many of these have in no way been correlated with definite chemical reactions, and the longer a thorough chemico-botanical investigation of our indigenous plants and the active principles they may contain is postponed, the longer will we continue to be faced by many unsolved problems like those recorded in the following pages.

Quite a variety of plant materials was sent to the Grahamstown Laboratory for examination in connection with the death of a Native at Mount Fletcher in 1905. Of the plants mentioned below none had been examined as to chemical or physiological properties. They are all very well worth thorough investigation. One was a species of *Tephrosia* administered to the deceased

* See page 13.

† R. Marloth: "The Chemistry of South African plants and plant products" (1913), 1.

shortly before his death. According to Bentley,* several species of *Tephrosia* are employed as fish poisons,*Tephrosia toxicaria* particularly. Bryant† declares *T. macropoda* and *T. diffusa* to be very poisonous. As for the pharmacological value of such plants as these, it has been suggested that *T. toxicaria,* which is supposed to resemble *Digitalis* in its action on the human system, may yet prove a useful substitute for the latter.

Another specimen received in connection with the above case was a plant administered to the deceased by a Native named Lekana, and stated by him to be *Mu-gumabela* or *um-Nungamabele*. The specimen was identified as *Rhus discolor* E. Mey., and it is worthy of mention that this plant is quite distinct from *Xanthoxylon capense* Harv., which Smith‡ identifies with *um-Nungamabele*. Several species of *Rhus* are more or less poisonous, and require very careful handling, as the liquids which they exude frequently cause violent erysipelatous inflammation.§ The particular species connected with the case under discussion is not described, but, in view of what has been stated above, it is significant that the District Surgeon, in his autopsy, found that

> The stomach and bowels were very much inflamed, and the membrane was covered with small hæmorrhages. There was also hæmorrhage in the kidneys, bladder, and lungs.

A third plant submitted in connection with this case was also said to have been administered by Lekana. This proved to be a species of *Thesium*, which is slightly astringent, and supposed to be harmless.

On examining the stomach of the deceased by the usual chemical process, no active plant principle of the ordinary type could be discovered, but the resin which separated during the process was found to yield very characteristic reactions with strong sulphuric acid. An effort was then made to discover whether a similar resin could be traced in any of the plants supposed to be connected with the case, and, as the quantities previously supplied had been insufficient for the purpose, fresh supplies were requested, with the result that a resin corresponding in every respect with that found in the deceased's stomach, was discovered in the *Thesium*, which was believed to be non-poisonous. The non-toxic character of this plant was subsequently confirmed by physiological experiments. The case was altogether one of those curious and inexplicable occurrences which have more than once happened where circumstantial evidence is entirely unsupported by the chemical analysis. Here a fatality was associated with a number of indigenous herbs, of which two are looked upon as powerful poisons, while a third is generally held to be harmless. It would have been thought

* Bentley: "A manual of Botany" (1887), 535.

† "Zulu Medicine and Medicine-men," in Annals of the Natal Museum '1909), **2** [1], 12.

‡ South African Materia Medica," 3rd ed. (1895), 44, 58, and 126.

§ Bentley: *Op. cit.*, p. 523.

that it was one of the former that had caused death, but analysis proves the presence only of the innocuous plant in the body of the deceased, and when, shaken in the belief that this plant is really as harmless as was supposed, tests are made by the investigator, he finds, somewhat to his surprise, that the theory of its harmless nature is fully confirmed.

In connection with a fatal poisoning case at Mqanduli, in 1907, various specimens of bulbs and roots were received for analysis, together with the stomach and intestines of the deceased woman. In the latter organs no trace of alkaloids or glucosides was detected, although a perceptible amount of resinous material was extracted, which will be referred to again. In the stomach were found vegetable particles closely resembling fragments of one of the bulbs submitted. One specimen of pieces of root and bulb,* on being tested for alkaloids and glucosides, gave negative results, but it contained a considerable amount of resin, *viz.*, 13 grains in 1¼ ounce of the material. This resin was, on further examination, found, by the reactions described below, to be identical with that obtained from the stomach. Another dried portion of a bulb, like the former, unnamed and unidentified, also contained the resinous body, already mentioned. A third specimen consisted of an entire bulb, fresh, and just commencing to sprout; it was referred to as *Tyumtyumana*.† and declared to be identical with the dried specimen. It had a distinctly acid reaction towards litmus, and possessed a bitter and very nauseous taste. From ¼ of an ounce thereof 7½ grains of a resinous body were extracted, but no alkaloid or glucoside. This resinous body was almost insoluble in cold water, and slightly soluble in ether, but dissolved more readily in chloroform, in alcohol, and in water which had been slightly acidulated. It was removed from acid solutions by immiscible solvents, dissolved in caustic alkalies with a darkening colour, and gave the following characteristic reactions:—

Concentrated sulphuric acid.	Yellow, brick-red; changing to dark brown.
Concentrated sulphuric acid, with trace of potassium dichromate.	Deep emerald green; changing to blue after one hour.
Concentrated nitric acid.	Bright yellow to brown.
Ferric chloride (aqueous).	Light brown precipitate.
Copper acetate (aqueous).	Bluish white precipitate; on shaking with ether, the latter is not coloured.
Copper acetate (alcoholic).	Greenish precipitate; supernatant liquid bluish.

* Stated to be fragments of the very bulb partaken of by the deceased.

† Dr. P. H. Walker, who gives details of this case in the *South African Medical Record* (1914), **12** [9], 142, identifies *Tyumtyumana* with *Bowiea volubilis* referred to on a former page of the present paper.

Lead acetate (aqueous).	White precipitate.
Lead acetate (alcoholic).	White precipitate.

To a full-grown healthy cat a quantity of the fresh bulb, representing three grains of the resin, was administered, together with six grains previously extracted from one half of the same bulb. The animal soon appeared very uncomfortable, began frothing at the mouth, and died in less than an hour. A *post-mortem* examination showed the organs to be normal, except the stomach and heart, the former of which contained a frothy mucus, while the right ventricle of the heart was distended with blood.

To a vigorous rat nearly two grains of the resin in aqueous solution were administered. Speedily it became ill, appeared to be in pain, and died within twelve hours. Examination revealed very similar appearances to those observed in the case of the cat: the stomach and intestines, moreover, were much congested. To another healthy rat a very much smaller quantity of the fresh root, powdered and mixed with 15 drops of water, was given. It also took ill shortly afterwards, and appeared to have spasms of severe pain, but after 24 hours began to recover.

It seemed from these experiments that the woman had died from the effects of taking the plant, which contained a soft bitter resin, acid in character, possessing a peculiar odour, and marked toxic properties. Fifteen grains of this resin were found in one bulb, and there was no doubt that, if administered in large doses, it would prove fatal.

Three years after the Mqanduli poisoning case just referred to, a similar case occurred at Kokstad. Amongst other articles, the medicine supplied to the deceased by the Kaffir "doctor" was examined. It possessed a herbal odour characteristic of decoctions compounded of *Tyumtyumana*, and a resinous body was extracted from it which proved identical, in the chemical reactions which it yielded, with that obtained from the Mqanduli *Tyumtyumana* root. This medicine measured 290 cc., of which 220 cc., after shaking, was examined by the Stas-Otto method for the presence of vegetable principles, with the result that a very small quantity (less than one-tenth of a grain) of a resinous body was extracted. At the most the total mixture in the bottle did not contain more than one-tenth of a grain of this toxic substance in solution. In addition thereto it also contained at least seven-tenths of an ounce of Epsom salts. To a healthy dog, Mr. Muller administered doses of ten and twenty cc. of this mixture on two consecutive days, with marked depressant effects. Thirty-five cc. was then administered in milk to another healthy and vigorous dog, with similar though more marked results. The animal recovered the next day, without any definite signs of purging. As the bulb used in compounding this medicine owes its irritant and toxic properties to the resin which it contains, it is possible that this medicine, after several months, may

to a certain extent lose its toxic nature, and become almost harmless, even in moderately large doses, as the resin seems more active in its effect when freshly prepared.

The blood from the deceased's heart appeared similar in colour and consistency to that resulting from partaking of the plant mentioned, and in the contents of the stomach vegetable fragments were found almost identical in structure with those separated from the medicine above referred to.

In Mafeking, during 1911, the wife of a native constable had been treated, with fatal results, by a Kaffir "doctor," who had administered to her an extract of boiled roots in doses of a tablespoonful three times daily, in order to produce pregnancy. From the stomach and kidneys of the deceased small quantities of a substance, possibly of an alkaloidal nature, were extracted, whch yielded the following reactions with the usual alkaloidal tests:—

Sonnenschein's reagent.	Very light greenish-yellow precipitate.
Scheibler's reagent.	Cream-coloured precipitate.
Schulze's reagent.	White precipitate.
Potassium cadmium iodide.	Orange precipitate.
Potassium bismuth iodide.	Light red precipitate.
Potassium zinc iodide.	Dark brown precipitate.
Iodine in potassium iodide.	Brownish orange precipitate.
Picric acid.	Yellow precipitate.
Platinum chloride.	White precipitate; changing to brownish.
Gold chloride.	White precipitate; changing to brownish.
Sulphuric acid, with potassium dichromate.	Dull purple; changing to dirty green.

During 1911, three cases of poisoning by indigenous plants, at Nqamakwe, were investigated. The first of these has been alluded to in connection with *Hæmanthus puniceus*. The circumstances of the second case were that the deceased, a Native, had for years been attended by European practitioners for pulmonary tuberculosis, and had ultimately called in a native herbalist, who, in order to cause vomiting, administered the roots which brought about the fatal result. A sample of the root supposed to have been administered to the deceased was found to contain a little inactive resin, an alkaloid, and a trace of a glucoside. The alkaloid reacted with all the common alkaloidal reagents, and also gave the following colour tests:—

Sulphuric acid.	Yellow to yellow-brown.
Nitric acid.	No reaction.
Vitali's reagent.	No reaction.
Fröhde's reagent.	Blue to violet, to cherry-red, and after 24 hours brown.

No effect was produced on a guinea-pig by the equivalent, in its active principle, of .7 of a grain of the root. Half that quantity, administered to a rat, caused vomiting within fifteen minutes, with ultimate recovery. A decoction of the root was then administered to the same two animals in the following proportions: to the rat, the equivalent of one-twentieth of a grain of the root, no effect being produced; to the guinea-pig, the equivalent of .35 of a grain of the root: in forty-five minutes spasmodic convulsions occurred, and death in two hours, the heart *post-mortem* being found dilated. The stomach of the deceased man contained an alkaloid yielding reactions identical with those of the root, and also causing violent emesis when administered to a rat. The rat, however, subsequently recovered. The active principle extracted from the stomach produced no effect on a guinea-pig, but the quantities thereof available for experiment were very small.

The third Nqamakwe case was also that of a man who had been treated, with fatal consequences, for an illness by a native herbalist. No toxic principles were discoverable, either in the stomach or in the unidentified plant materials supposed to have been the cause of death.

In a case which originated at Tsomo, during the same year, no poisonous principles were found in the native "medicines" (including two pieces of roots) which the deceased was supposed to have taken, but from the stomach there was extracted an alkaloid soluble in ether and chloroform, and capable of extraction by ether from an acidified solution. This responded to the usual alkaloidal tests, and gave the following colour reactions, which are not characteristic of any known alkaloid:—

Sulphuric acid.	Yellow; changing to orange, and then to brown.
Nitric acid.	No reaction.
Fröhde's reagent.	Green.
Vitali's reagent.	No reaction.
Erdmann's reagent.	Violet; changing into red.

One-twelfth of the quantity of unknown alkaloid found in the stomach had a violent emetic and purgative action on a guinea-pig, the animal expiring within twenty hours, with intense inflammation of the digestive tract, and particularly of the stomach. The same symptoms and lesions occurred in the case of a rat, which recovered from a first dose equivalent to one-twenty-fourth of the stomach, but died within 24 hours of a second similar dose administered after an interval of two days. From a small quantity of unknown roots received two months later, an alkaloid was extracted giving precisely identical colour reactions with those just described, and causing vomiting and death in two guinea-pigs, within 30 and 60 minutes respectively, after administration of one gramme of the plant in each case.

and in 20 hours in the case of a rat to which half a gramme had been administered. The poisonous nature of the roots, as well as their identity with that taken by the Tsomo Native, was thus established, but just what the particular genus and species of plant was from which the roots in question were derived, remained in absolute obscurity, and an important point to be remembered in connection with these cases of unestablished identity is, as Dr. Walker points out,* that a loophole is thus left for poisoners to get off scatheless.

In connection with a fatal case that occurred at Kimberley in 1912, it became evident that death had been caused by a poisonous bulb, but to identify that bulb was an impossibility. A decoction had been administered to the deceased, and this having been first examined, pieces of a bulb were found therein. The material vomited by the deceased contained fragments of a bulb microscopically identical with that found in the decoction. This material was discovered to contain a glucoside similar in properties and reactions to those of a bulb found in the "medicine chest" of the Kaffir "doctor" who administered the decoction to the deceased. In the deceased's stomach several pieces of the bulb already referred to were found, and the stomach contents proved to contain traces of a glucoside identical with that extracted from the bulb in the "medicine chest." This "medicine chest" was a boot box, containing a heterogeneous collection of bulbs, roots, barks, etc., many being well-known Kaffir remedies. Of one particular bulb there were about eighteen specimens, weighing from about eight to sixteen grammes each. As already stated, sections of these bulbs were microscopically examined, and proved identical in character with those found in the decoction, and in the deceased's vomit and stomach. Two of the bulbs, weighing together 28.9 grammes, were finely cut up, and an infusion made with distilled water, the liquid being finally filled up to 100 cc., and filtered. Of the clear filtered infusion, 5 cc. was administered to a healthy and vigorous dog, aged about eight to ten months. It remained well and lively for about two hours; half an hour later it vomited, remaining ill through the subsequent night. It appeared to recover about 20 hours after administration of the infusion, and three hours later was apparently once more quite well and lively. On the fourth day after this, 10 cc. of the same infusion was injected hypodermically into the same dog. Two hours later the dog vomited repeatedly, passed quantities of mucus *per anum,* and five hours after injection was much worse and practically moribund. It died during the night. The Acting Government Pathologist reported:—

> All organs healthy to the naked eye. Heart—right side dilated and filled with *post-mortem* clot. Left ventricle contracted and empty.

The drug appears to have a very powerful effect on the gastro-intestinal canal, and may therefore finally bring about

* *op. cit.,* p. 141.

cardiac failure, but it cannot be assumed that it is primarily a heart-poison.

The remaining sixteen small bulbs, weighing in all 96.2 grammes, were finely cut up and treated with alcohol for several days. The clear filtrate was found to contain a resin and a glucoside, but in very small quantity; apparently .04 per cent. in the air-dried bulbs. From this the following reactions were obtained:—

Water.	Almost insoluble.
Very dilute hydrochloric acid.	Difficultly soluble.
Alcohol.	Very soluble.
Chloroform.	Readily soluble.
Ammonia or sodium hydroxide.	No precipitate.
Concentrated sulphuric acid.	Yellowish-brown to vandyke brown.
Concentrated sulphuric acid, with potassium dichromate.	Bright yellow to bluish green.
Concentrated sulphuric acid, with nitric acid fumes.	Yellowish brown to yellow.
Nitric acid.	Bright canary yellow; changing immediately to violet, and then gradually to yellow.
Fuming nitric acid.	Transient deep purple to yellow.
Vitali's reagent.	Yellowish brown.
Gold chloride.	Nil.
Platinum chloride.	Nil.
Picric acid.	White curdy precipitate.
Phosphomolybdic acid.	Pale greenish precipitate.
Mercuric chloride.	Nil.
Scheibler's reagent.	Crystalline yellowish precipitate.
Schulze's reagent.	Pale yellowish precipitate.
Potassium zinc iodide.	Light orange precipitate.
Potassium cadmium iodide.	Bright orange precipitate.

The infusion and tincture of the bulb possessed a peculiar aromatic odour, and this odour was again developed when dissolving the impure glucoside in a weak solution of an acid. The test with nitric acid was very marked, and this reaction was made special use of in detecting the presence of the active principle in the final residues obtained from the vomit and the stomach contents.

A Native at Idutywa, during 1911, was supposed to have been poisoned by an unidentified bulb, a specimen of which (weighing 84.5 grammes) was submitted for analysis. Extracts of the bulb administered to rats caused tremors, straining, and vomiting, with subsequent recovery. Administered to two guinea-pigs, portions of the extracts speedily resulted in

convulsive tremors, gasping, and death, the fatal termination supervening in ten minutes in one case, and in the other—with half the dose of the former—in 2¼ hours. *Post-mortem* examination was made in both cases at the Veterinary Institute, and the report showed that in both guinea-pigs there were hyperæmia and œdema in respect of the lungs, while the liver was congested and swollen, and both kidneys congested. It was also noted, in regard to the second guinea-pig, that both kidneys were dark in colour, that the left heart was empty of blood, and that there was slight hyperæmia of the small intestines.

Chemical examination of the bulb led to the separation of an alkaloid in very small quantity, and of a glucoside, bitter in taste, insoluble in ether, and readily decomposed by warm dilute acid into glucose and a resinous substance. A little of this glucoside administered to a rat gave rise to symptoms similar to those above described. The difference between the rats and the guinea-pigs was that, while the rats vomited most of the material administered to them, the guinea-pigs retained it and died.

When the examination had proceeded thus far, the stomach and other viscera of the deceased were received in the laboratory. From the stomach was extracted a glucoside soluble in water and alcohol, but insoluble in ether, and from the other organs a very small quantity of the same glucoside. This glucoside also proved to be toxic, and a quantity representing one-twentieth of the stomach caused death in guinea-pigs, and brought about symptoms of poisoning in a rat, which, however, vomited and then recovered. In so far as the quantity of unidentified plant material permitted of conclusions, the glucoside found in the stomach was identical with that occurring in the bulb previously analysed.

A case that occurred at Herschel, in 1912, and investigated in the Grahamstown Laboratory, was another in which an unidentified bulb was concerned. A native "doctor" had administered a quantity of an infusion or decoction to an old man, aged 70 or 80 years. Vomiting and purging ensued, and death soon followed. *Post-mortem* examination revealed old pleurisy and bronchitis; the heart was senile and flabby, and tapeworm was present. The medical opinion was to the effect that excessive vomiting and purging had caused collapse, the deceased having been frail, and his health impaired by lung trouble. The unknown bulb supposed to be the cause of death was found, upon chemical examination, to contain .3 per cent. of a glucoside, with a very small quantity of an alkaloid. About ten grains of this bulb, administered to a guinea-pig, caused its death in ninety minutes. The symptoms were convulsive breathing, spasms, and slowing of the heart. *Post-mortem* examination showed the heart engorged, and hyperæmia of the lungs, liver, and kidneys, death being due to heart failure. A rat was then dosed with a

small quantity of the alcoholic extract of the bulb, and within half an hour showed signs of straining, with twitching of the limbs, but soon recovered. Five grains of a second bulb caused the death of a guinea-pig in 3½ hours, producing symptoms and lesions similar to those caused by the bulb first tested. The contents of the deceased man's stomach consisted of one fluid ounce of a thin gruel-like fluid, and it yielded a trace of an alkaloid, and a very small amount of a glucoside. Dr. Lewis tested one half of the active principles extracted from the stomach on a guinea-pig. In ninety minutes the animal showed uneasiness and twitching of the limbs, followed by spasms. It subsequently recovered. The bulbs seemed to contain a poison acting directly on the heart, and dangerous in large doses even to healthy human beings. The tests on the stomach contents, though seeming to indicate the presence of a small quantity of a similar substance, were not decisive enough to warrant a definitely affirmative pronouncement.

In connection with the death of a native woman at Mqanduli in 1912, only the organs of the deceased were received, not only unaccompanied by the actual plant supposed to have caused death, but even without any plant substances whatever. The organs were chemically examined, with a view to ascertaining the presence of any toxic plant materials, but none such were found. A substance was, however, isolated from the viscera, giving the following reactions:—

Ether.	Soluble.
Dragendorff's reagent.	Dark brown precipitate.
Sonnenschein's reagent.	White precipitate.
Scheibler's reagent.	Slight white precipitate.
Ferric chloride.	Greenish-black colouration.
Sulphuric acid and potassium dichromate.	Green colouration.
Sulphuric acid.	No reaction.
Nitric acid.	No reaction.
Fröhde's reagent.	No reaction.
Lead acetate.	Not precipitated.

Mr. Sinclair tested this substance physiologically on guinea-pigs, but the animals seemed to suffer no ill effects.

Two fatalities at Mount Ayliff were investigated in the Grahamstown Laboratory during 1912, and in neither of these was identification of the plant poison possible. In connection with the first one a native herbalist had reported that a syphilitic woman, who had consulted him, had been subsequently found by him hanging from a tree and quite dead. He declared that he had given her no medicine, but it was evident from the *post-mortem* examination that death had been slow, and was certainly not the result of hanging. The organs of the deceased were found to contain a small quantity of an alkaloid, and extracts thereof administered to two guinea-pigs caused death respec-

tively in three and 24 hours, *post-mortem* examination of the animals showing congestion of the liver in both and slight inflammation of the intestine in the guinea-pig that died in three hours. It was therefore concluded that the organs of the deceased contained a vegetable poison.

Another Mount Ayliff case was administered by Mr. Sinclair in the same year, and again the bulbs that acted fatally were unidentified and indefinite. Here, too, the actual medicines administered to the deceased were unprocurable, and, as in other cases, the specimens of bulbs submitted were only stated to be similar to that with which the deceased woman was supposed to have been dosed. Practically all that was worth doing under the circumstances was to test the bulbs physiologically, and so a dose of about 15 grains was administered to a guinea-pig, and the animal died about four hours later. To each of two older guinea-pigs about eight grains were given, and in both cases they sickened in about two hours, and died twelve hours after being dosed. *Post-mortem* examination in all three cases showed hyperæmia of the lungs, and of the small intestine. The District Surgeon, however, in his autopsy upon the deceased woman, found her lungs to be healthy, and a chemical analysis of her organs failed to disclose the presence of any toxic plant substance.

A native woman who died at Tsolo in 1908, about three hours after partaking of a decoction prepared by a Kaffir "doctor," had commenced vomiting and purging very soon after drinking the liquid, but as emetics and other remedies had been administered in the interval between partaking of the drug and the fatal issue, the symptoms may thus have been complicated. After death the stomach was found to be greatly inflamed, and the spleen enlarged and ruptured; the liver and lungs were greatly congested, and the latter engorged with blood. The veins were also engorged, and so was the right side of the breast, while hæmorrhage had occurred in the abdominal cavity. The rupture of the spleen, in the opinion of the District Surgeon, had been caused by excessive vomiting.

It would assist the analyst greatly, and further scientific and practically useful discovery, if fuller information were forthcoming with regard to the plants used in cases like this. In the case now under discussion a mere bulb—destitute of leaves and flowers—was all that was received in the Laboratory for analysis in connection with the case. For over a month an unsuccessful attempt was made to get it to grow, with a view to securing its identification, which was impossible in its meagre condition. Obviously, while legal proceedings were pending, there was little time for investigation, and so the attempt had to be abandoned. I mention this in order to emphasise the importance of undertaking such investigations *at leisure,* and not when pressed by criminal trials. All that could be learnt regarding the bulb was that its native name is *Usigagana* (? *isi-Gagana*), and endeavours

were unsuccessfully prosecuted for over nine months to procure a specimen with leaves and flowering stalk complete for botanical identification. There remained apparently nothing for it but to confine investigation to practical tests of the bulb's toxic properties: a decoction was therefore prepared from six grammes of the bulb, and administered to a healthy dog, ten to twelve months old. Vomiting, accompanied by severe straining, set in within five minutes, and continued until death, which supervened within half an hour. The heart was found to be distended, and engorged with blood, and the muscular tissue was in a flabby condition. Another portion of the bulb could now be examined chemically: this was accordingly done, and the bulb was found to contain an active principle, to all appearances a glucoside, which, on closer investigation, proved to be chemically identical with one previously met with in a bulb from the same district.*

From the stomach contents, by the Stas-Otto method, a substance was extracted which gave reactions identical with those of the bulb, but was too small in quantity to permit of any physiological tests being made. The active principle in the plant, which, it is hoped, may yet be botanically identified, is undoubtedly powerfully poisonous, producing emesis and purging, followed by collapse and death, and the fatal issue may result either from exhaustion consequent upon violent and continued straining, or from paralysis of the heart in diastole.

No plant belonging to the sub-order *Melanthaceæ*, or *Colchicaceæ*, which occurs throughout South Africa, and contains larger or smaller quantities of the alkaloid colchicine, of which a quantity varying from one grain to three grains proves fatal, has yet been examined in the Cape Government Laboratories, but such a case was investigated by Prof. Hahn in 1892. It occurred at Mount Frere, where a native died after receiving an injection of herbs belonging to a species of the sub-order named. Dr. Hahn estimated the amount of colchicine in the injection at four and one-sixth grains. In the stomach and contents no trace of colchicine or any other poison could be found, but from the intestines one grain and two-thirds of the alkaloid was extracted.

Strychnine, although a plant poison, scarcely comes under the category of plant-poisons peculiar to South Africa, and therefore my excuse for mentioning a case of strychnine poisoning is the astonishing way in which the victim recovered. One of the most remarkable recoveries from strychnine poisoning on record was that of Dr. W. T. Harris, at King Williamstown, related in Taylor's "Principles and Practice of Medical Jurisprudence."* The following, which I append as my closing example of Cape poison cases, is in one way more remarkable, although unfortunately lacking in details. It occurred in the Division of Sutherland in 1912. The attendant upon the victim had determined

* See "Notes regarding South African Pharmacology," p. 130.

to get rid of him, so as to acquire certain of his possessions, and, previously to partaking of coffee together in the open veld, had so liberally dosed his sugar with strychnine that, when it subsequently arrived in the laboratory, it appeared plentifully studded with the pink-dyed crystals. A single teaspoonful of the sugar (weighing 4.86 grammes) taken at random from the 8¼ ounces submitted for analysis, contained 1.82 grain of strychnine, or not far short of 2½ per cent. It appears that the victim, suspecting nothing, drank some of the coffee before noticing that it had a bitter taste; but when it was noticed and the situation realised, he immediately commenced smoking as energetically as possible, and also swallowed as much pipe oil as he could. This did not avert the severe symptoms of strychnine poisoning, which speedily set in, although it did avert a fatal termination. Three months later, when the trial of the accused took place, the characteristic spasms were still very noticeable while the victim was in attendance at the Circuit Court for the purpose of giving evidence.

I have come to the end of my record of poisoning cases, and I must confess that it is at best a record of casual and perfunctory tinkering with a very important branch of chemical investigation. For nearly twenty years I have been most persistent in urging that there should be earnest and systematic research into what is a veritable treasure-house of pharmacology, and all the while there has not been one branch of the laboratories' work on a more unsatisfactory footing. That there are in this country numerous indigenous plants containing poisonous or pharmaceutically valuable principles hitherto unknown to science is a fact beyond all doubt, and, as a matter of pure original research, quite apart from its utilitarian value, it would be both interesting and instructive to make a series of investigations into the nature of such plants and their unknown constituents, but for investigations like these far more time would be needed than can be afforded when a particular case of death or illness is under enquiry. I think that every reader of these notes will admit that some of the results recorded are of the nature of loose ends, which it would be of considerable scientific interest to follow up if one only had the leisure for it. There is abundant scope for enquiry, but all too little opportunity to take advantage of it. In fact there is scarcely a field of chemical research in connection with South Africa that is vaster in the promise it holds out to explorers, and yet is so utterly unexplored. And this in view of the fact that to the practical man the subject is as coldly utilitarian as he could wish, for there is no doubt that indigenous plants, at present uninvestigated, frequently play an active part in mysterious deaths amongst the natives, and the circumstances associated with these fatalities are consequently such as to render the production of the *corpus delicti*, within the limited time required by the law courts, a matter of great difficulty, if, in fact, not actually

* 5th ed. (1905) **2**, 819-821. See also *South African Medical Journal* (1895), 341.

impracticable. It would be well if, in the Government's agricultural laboratories—for the matter is as much one of stock as of human poisoning—one or more chemists were specially delegated to investigate the problems arising out of the many poisoning cases that occur, particularly in the native territories. Under present circumstances, whenever a poisoning, or suspected poisoning, case occurs, problems have to be grappled with at short notice, and in a hurried manner, which should have been carefully investigated at leisure months beforehand. If this were done the scientific recognition of the poisons found would rest on more assured ground than the hurried examinations which now are the side-issues of the law courts, and human beings charged with the crime of murder or culpable homicide would not depend for life or liberty on casual investigations which must be *made* to do, so long as more thorough research is regarded as merely academic. At present it is the very meagreness of the knowledge that we have acquired regarding the plant drugs and plant poisons of the country that makes the further pursuit of this knowledge seem academic. It is the very incidental character that these investigations are compelled to assume that militates against results of enduring usefulness being arrived at. Matters of this kind should be dealt with because of their intrinsic, scientific, and practical value, and not as mere side-issues of the legal proceedings against some Kaffir "doctor."

Until we make a special study of the many poisonous plants indigenous to the country, Kaffir "doctors" will no doubt continue to kill their "patients" with plants of whose poisonous character we, the civilised and cultured, are unaware, and with far less restraint from the law than public safety demands, and this simply because, in many cases, we do not know enough about the poisons employed either to enable chemical tests to be applied with unerring accuracy, where those poisons have been administered with fatal result, or to ensure the use of reliable antidotes so as to avert such result. Although the lives at stake are generally those of natives, the principle is important enough to warrant more thorough-going investigation than it now receives.

It is, moreover, in my opinion, a distinct reflection on the status of science in South Africa that the only serious effort that is being made to-day to investigate the chemical composition of South African plants is being made in the laboratories of private manufacturing firms 6,000 miles away, and by chemists who have to import into England by the hundredweight, for that special purpose, plants that they consider it worth while to undertake so much additional trouble to examine, while we, with the wealth of the country's flora at our very doors, are so little alive to its capabilities, and so apathetic with regard to the industrial possibilities latent therein, that we are not even ashamed at our inaction.

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